Hazardous Chemicals Desk Reference

Second Edition

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VNR VAN NOSTRAND REINHOLD
New York

idizers. To fight fire, use alcohol foam, , water spray, fog, dry chemical. When d to decomposition it emits acrid and irritatumes.

V750 CAS: 10108-56-2 **HR: 3**
JTYL CYCLOHEXYL AMINE
$C_{10}H_{21}N$ mw: 155.32

P: Flash p: 200°F (OC), d: 0.8, bp: 210°.

ETY PROFILE: A poison by ingestion. erately toxic by skin contact. A skin irritant. bustible when exposed to heat or flame. ght fire, use alcohol foam. When heated composition it emits toxic fumes of NO_x.

000 CAS: 61925-70-0 **HR: 3**
-tert-BUTYL CYCLOHEXYL)-3,3-IENYL PROPYLAMINE ROCHLORIDE
$C_{25}H_{25}N \cdot ClH$ mw: 375.97

: MG 18037

ETY PROFILE: A poison by intraperitoneal . Moderately toxic by ingestion. When d to decomposition it emits very toxic s of HCl and NO_x.

000 CAS: 94-80-4 **HR: 3**
YL DICHLOROPHENOXYACETATE
$C_{12}H_{14}Cl_2O_3$ mw: 277.16

S: BUTYL 2,4-D * BUTYL (2,4-DICHLOROPHE-)ACETATE * 2,4-D BUTYL ESTER * BUTYL 2,4-D * (2,4-DICHLOROPHENOXY)ACETIC ACID, L ESTER * ESSO HERBICIDE 10 * FER- * LIRONOX * SHELL 40

ISENSUS REPORTS: IARC Cancer Re- : Animal Inadequate Evidence IMEMDT 11,77

ETY PROFILE: Poison by an unspecified . Moderately toxic by ingestion and possiother routes. Experimental teratogenic and ductive effects. Questionable carcinogen. erbicide. When heated to decomposition its toxic fumes of Cl^-.

500 CAS: 51003-83-9 **HR: 3**
BUTYL-3-DIMETHYLAMINO-5,6-HYLENEDIOXYINDENE ROCHLORIDE
$C_{16}H_{21}NO_2 \cdot ClH$ mw: 295.84

SYNS: 6-BUTYL-5-DIMETHYLAMINO-5H-INDENO(5,6-d)-1,3-DIOXOLE HYDROCHLORIDE * bu-MDI

SAFETY PROFILE: A poison by intraperitoneal and intravenous routes. When heated to decomposition it emits very toxic fumes of NO_x and HCl.

BRE500 CAS: 88-85-7 **HR: 3**
2-sec-BUTYL-4,6-DINITROPHENOL
mf: $C_{10}H_{12}N_2O_5$ mw: 240.24

PROP: Crystals. Vap d: 7.73.

SYNS: ARETIT * BASANITE * BNP 30 * BUTAPHENE * CALDON * CHEMOX GENERAL * CHEMOX P.E. * DINITRO * DINITRO-3 * 4,6-DINITRO-2-sec.BUTYLFENOL (CZECH) * 2,4-DINITRO-6-sec-BUTYLPHENOL * 4,6-DINITRO-o-sec-BUTYLPHENOL * 4,6-DINITRO-2-sec-BUTYLPHENOL * DINITROBUTYLPHENOL * 4,6-DINITRO-2-(1-METHYL-N-PROPYL)PHENOL * 2,4-DINITRO-6-(1-METHYL-PROPYL)PHENOL (FRENCH) * DINOSEB * DINOSEBE (FRENCH) * DN 289 * DNBP * DNOSBP * DNSBP * DOW GENERAL * DOW GENERAL WEED KILLER * DOW SELECTIVE WEED KILLER * ELGETOL * ELGETOL 318 * ENT 1,122 * GEBUTOX * HEL-FIRE * KILOSEB * 6-(1-METHYL-PROPYL)-2,4-DINITROFENOL (DUTCH) * 2-(1-METHYLPROPYL)-4,6-DINITROPHENOL * 6-(1-METIL-PROPIL)-2,4-DINITRO-FENOLO (ITALIAN) * NITROPONE C * PHENOTAN * PREMERGE * PREMERGE 3 * RCRA WASTE NUMBER P020 * SINOX GENERAL * SPARIC * SPURGE * SUBITEX * UNICROP DNBP * VERTAC DINITRO WEED KILLER * VERTAC GENERAL WEED KILLER * VERTAC SELECTIVE WEED KILLER

CONSENSUS REPORTS: EPA Genetic Toxicology Program. EPA Extremely Hazardous Substances List.

SAFETY PROFILE: A poison by ingestion, inhalation, skin contact, subcutaneous, intraperitoneal, and possibly other routes. Experimental teratogenic and reproductive effects. A severe eye irritant. Questionable carcinogen with experimental tumorigenic data. Mutation data reported. An herbicide. When heated to decomposition it emits toxic fumes of NO_x.

BRF500 CAS: 50-33-9 **HR: 3**
4-BUTYL-1,2-DIPHENYL-3,5-DIOXO PYRAZOLIDINE
mf: $C_{19}H_{20}N_2O_2$ mw: 308.41

SYNS: ALINDOR * ALKABUTAZOI RIN * ANERVAL * ANPUZONE * ANUSPIRAMIN * ARTIZIN * A * ARTROPAN * AZDID * AZOBU LID * BENZONE * BETAZED * * B.T.Z. * BUSONE * BUTACOM * BUTACOTE * BUTADION * BU * BUTAGESIC * BUTALAN * BU * BUTALIDON * BUTALUY * BU * BUTAPIRAZOL * BUTAPYRAZOLI BON * BUTARTRIL * BUTARTRIN ZINA * BUTAZOLIDIN * BUTAZO ZONE * BUTE * BUTIDIONA * SIMPLE * BUTONE * BUTOZ * PHENYLPYRAZOLIDINE-3,5-DIONE * PHENYL-3,5-PYRAZOLIDINEDIONE * * BUVETZONE * BUZON * CHE * FA-192 * DIGIBUTINA * DIOSS * 3,5-DIOXO-1,2-DIPHENYL-4-N-BUTYL * 3,5-DIOXO-1,2-DIPHENYL-4-N-BUTYL * 3,5-DIOXO-1,2-DIPHENYL-4-N-BUTYL * DIOZOL * DIPHEBUZOL * DIPI TAZONE * 1,2-DIPHENYL-4-BUTYL-3, RAZOLIDINE * 1,2-DIPHENYL-4-BUTY LIDINEDIONE * 1,2-DIPHENYL-3,5-DI RAZOLIDINE * 1,2-DIPHENYL-2,3-DIC PYRAZOLINE * ECOBUTAZONE * * EQUI BUTE * ERIBUTAZONE * * FBZ * FEBUZINA * FENARTIL SAN * FENIBUTAZONA * FENIBU BUTAZONA * FENILBUTINE * FE * FENOTONE * FENYLBUTAZON * IA-BUT * INTALBUT * INTRA * IPSOFLAME * KADOL * LINGI GESIC * MEPHABUTAZONE * ME * NADAZONE * NADOZONE * N * NEO-ZOLINE * NOVOPHENYL * * PHEBUZIN * PHEBUZINE * PH * PHENBUTAZOL * PHENOPYRINE TAZ * PHENYLBUTAZON (GERMAN) TAZONE * PHENYLBUTAZONUM * BUZON * PIRARREUMOL 'B" * PR * PYRABUTOL * PYRAZOLIDIN * * REUDO * REUDOX * REUMAS ZIN * REUMAZOL * REUPOLAR * RUBATONE * R-3-ZON * SCAN * SCHEMERGIN * SHIGRODIN * * TETNOR * TEVCODYNE * THE * TICINIL * TODALGIL * USAF (* UZONE * VAC-10 * WESCOZOI PHEN * ZOLIDINUM * ZORANE

CONSENSUS REPORTS: IAR view: GROUP 3 IMEMDT 7,31

TOXICOLOGY

Nitroaromatic compounds are highly toxic to humans and animals. Most nitrophenols and nitrocresols are well absorbed from the gastrointestinal tract, across the skin, and by the lung when fine droplets are inhaled. Fatal poisonings have occurred as a result of dermal contamination. Except in a few sensitive individuals, they are only moderately irritating to the skin and mucous membranes.

Nitrophenols and nitrocresols undergo some biotransformation in humans, chiefly reduction (one nitro group to an amino group) and conjugation at the phenolic site. Although nitrophenols and metabolites appear consistently in the urine of poisoned individuals, hepatic excretion is probably the main route of disposition. Elimination is slow: residence half-life in humans is 5-14 days. Blood and tissue concentrations tend to increase progressively if an individual is substantially exposed on successive days.

Nitrophenols and nitrocresols are toxic to the liver, kidney, and nervous system. The basic mechanism of toxicity is stimulation of oxidative metabolism in cell mitochondria, by interference with the normal coupling of carbohydrate oxidation to phosphorylation (ADP to ATP). The nitrophenols are more active as uncouplers than chlorophenols. Increased oxidative metabolism leads to hyperthermia, tachycardia, and dehydration, and in time, depletes carbohydrate and fat stores. Most severe poisonings from absorption of these compounds have occurred in workers laboring in hot environments. Hyperthermia and direct action on the brain cause cerebral edema, manifest clinically as a toxic psychosis and sometimes convulsions. Liver parenchyma and renal tubules show degenerative changes. Albuminuria, pyuria, hematuria, and azotemia are prominent signs of renal injury.

Neutropenia has occurred in humans following heavy exposure to dinitrophenol. Cataracts occur in laboratory animals given nitrophenols, and have occurred in humans, both has a result of ill-advised medicinal use and as a consequence of occupational exposure. Cataract formation is sometimes accompanied by glaucoma.

6. Save a sample of emesis or initial gastric washings for chemical analysis.

5. During convalescence, administer a high-calorie, high-vitamin diet to restore body fat and carbohydrate.

6. Discourage subsequent contact with the toxicant for 4-8 weeks (depending on severity of poisoning) to allow full restoration of normal metabolic processes.

- **Adults and children over 12 years:** 5-10 mg. Repeat if necessary to a maximum of 30 mg.

- **Children under 12 years:** 0.25-0.40 mg/kg body weight. Repeat if necessary to a maximum of 10 mg for children 5-12 years and to a maximum of 5 mg for children 30 days to 5 years.

- Diazepam can be given by deep intramuscular injection if intravenous administration is not possible.

- **CAUTION:** Be prepared to assist pulmonary ventilation mechanically if respiration is depressed, to intubate the trachea if laryngospasm occurs, and to counteract hypotensive reactions.

7. Hemodialysis has not proven to be effective in poisonings by phenolic substances. Forced diuresis is of little or no benefit in reducing body burden. There has been insufficient testing of hemoperfusion to establish its value in accelerating elimination of phenols.

4. If nitrophenol or nitrocresol has been **ingested** in a quantity sufficient to cause poisoning, the stomach and intestine must be emptied and measures taken to limit absorption of residual toxicant. The effectiveness of induced emesis and gastric lavage in removing toxicant from the stomach diminishes rapidly with the passage of time.

1. **If** ingestion occurred within the last few hours, and **if** the patient is **fully alert** , give **Syrup of Ipecac**, followed by 1-2 glasses of water, to induce vomiting. The dosage of Syrup of Ipecac for adults and children over 12 years is 30 ml; the dosage for children under 12 years is 15 ml.

   - **CAUTION: Observe** the patient closely **after** administering **Ipecac**. Position the patient in left lateral decubitus, head below the level of the stomach. If consciousness level declines or if vomiting does not occur in 30 minutes, proceed immediately to **protect the airway** , then **intubate, aspirate, and lavage** the stomach (see below).

2. **If** the patient is **not fully alert** when first examined, proceed directly to **protect the airway** , insert a large bore orogastric tube, and empty the stomach by **aspiration and lavage** with a slurry of **activated charcoal** . See Organophosphate Insecticides, Treatment, Section 6.

3. Following emesis or lavage, administer **activated charcoal and cathartic** by ingestion or by orogastric tube, as recommended in the above reference.

4. If several hours have elapsed since ingestion, and if the patient is fully alert, **administer activated charcoal and cathartic orally** .

5. **Repeated** administration of **activated charcoal** at half or more the initial dosage every 2-4 hours may be beneficial.

   - **CAUTION:** Catharsis may lead to dehydration and electrolyte disturbances, particularly in children. Fluid balance and serum electrolytes should be monitored. There may be some advantage in giving repeated doses of cathartics to adults, but caution must be exercised in children. Administration of cathartic should stop when a charcoal stool appears.

TREATMENT OF NITROPHENOL OR NITROCRESOL POISONING

1. If poisoning has been caused by contamination of body surfaces, **bathe and shampoo** contaminated **skin and hair** promptly and thoroughly with soap and water, or with water alone if soap is not available. Wash the chemical from skin folds and from under fingernails. **Contaminated clothing** should be promptly removed, bagged, and not returned until it has been thoroughly laundered. Contaminated leather shoes should be discarded. The possibility that pesticide has contaminated the inside surfaces of gloves, boots, and headgear should be kept in mind.

2. **Flush** chemical from **eyes** with copious amounts of clean water. Obtain medical attention if irritation or other injury persists.

3. Systemic poisoning must be treated by controlling body temperature, providing oxygen, maintaining hydration, and relieving agitation.

    1. **Reduce elevated body temperature by physical means.** Administer sponge baths and cover victim with cool blankets. In fully conscious patients, administer cold, sugar-containing liquids by mouth as tolerated.

    2. **Do not** administer atropine, aspirin, or other salicylates to control hyperthermia. These agents appear likely to enhance the toxicity of phenolic substances. Neither the safety nor the effectiveness of other antipyretics has been tested.

    3. Administer **oxygen** continuously by mask to minimize tissue anoxia.

    4. Unless there are manifestations of cerebral or pulmonary edema or of inadequate renal function, administer **intravenous fluids** to restore hydration and support physiologic mechanisms for heat loss and toxicant disposition. Monitor serum electrolytes, adjusting IV infusions to stabilize electrolyte concentrations. Follow urine contents of albumin and cells, and keep an accurate hourly record of intake/output to forestall fluid overload if renal function declines.

        - **CAUTION:** In the presence of cerebral edema and/or impaired renal function, intravenous fluids must be administered very cautiously to avoid increased intracranial pressure and pulmonary edema.

    5. In severe poisonings, monitor pulmonary ventilation carefully to insure adequate gas exchange, and monitor cardiac status by ECG to detect arrhythmias. The toxicant itself and severe electrolyte disturbances may predispose to arrhythmias and myocardial weakness.

    6. To reduce production of heat in the body, **control agitation** and involuntary motor activity with sedatives. **Diazepam** or other benzodiazepine should be effective, although use of these drugs in nitroaromatic poisoning has not been reported. If diazepam is chosen, administer **slowly** , intravenously.

        - Dosage of **DIAZEPAM**:

CONFIRMATION OF POISONING

Unmetabolized nitrophenols and nitrocresols can be identified spectrophotometrically, or by gas-liquid chromatography, in the serum and urine at concentrations well below those that have been associated with acute poisonings. Blood analysis is useful in confirming the cause of poisoning, but has little value in monitoring progress or predicting outcome. If poisoning is probable, **do NOT await confirmation** before commencing treatment, but save urine and blood specimens in the event confirmation is necessary later on.

SYMPTOMS AND SIGNS OF POISONING

Yellow staining of skin and hair often signify contact with a nitroaromatic chemical. Staining of the sclerae and urine indicate absorption of potentially toxic amounts. Profuse **sweating, headache, thirst, fever,** confusion, malaise, and lassitude are common early symptoms of poisoning. Warm, flushed skin, tachycardia, and tachypnea indicate a serious degree of poisoning. **Restlessness**, apprehension, anxiety, manic behavior, or unconsciousness reflect cerebral injury. **Convulsions** signify an immediate life-threatening intoxication. Labored breathing and cyanosis are consequences of the stimulated metabolism and tissue anoxia. Weight loss occurs in persons continually exposed to relatively low doses if nitrophenols or nitrocresols.

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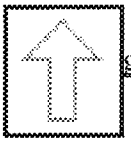

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Nitrophenolic and Nitrocresolic Herbicides

Donald P. Morgan, M.D., Ph.D.

These agents have many uses in agriculture worldwide: herbicides (weed-killing and defoliation), acaricides, nematocides, ovicides, fungicides. Relatively insoluble in water, most technical products are dissolved in organic solvents and are formulated for spray application as emulsions. There are some wettable powder formulations.

Commercial Products: Dinitrophenol (Chemox PE), dinitrocresol (DNOC, DNC, Chemsect DNOC, Elgetol 30, Nitrador, Selinon, Sinox, Trifocide), dinoseb (DNBP, dinitro, Basanite, Caldon, Chemox General, Chemox PE, Chemsect DNBP, Dinitro, Dinitro-3, Dinitro General, Dynamyte, Elgetol 318, Gebutox, Hel-Fire, Kiloseb, Nitropone C, Premerge 3, Sinox General, Subitex, Unicrop DNBP, Vertac, Dinitro Weed Killer 5, Vertac General Weed Killer, Vertac Selective Weed Killer), dinoseb acetate (Aretit), dinoseb methacrylate (binapacryl, Morocide, Acricid, Ambox, Dapacryl, Endosan, FMC 9044, Hoe 002784, Morrocid, NIA 9044), dinosulfon, dinoterbon, dinoterb acetate, dinoterb salts, dinosam (DNAP, Chemox General), dinoprop, dinocap (Crotothane, Karathane), dinobuton (Acrex, Dessin, Dinofen, Drawinol, Talan), dinopenton.

- TOXICOLOGY
- SYMPTOMS AND SIGNS OF POISONING
- CONFIRMATION OF POISONING
- TREATMENT OF NITROPHENOL OR NITROCRESOL POISONING

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